A STUDY ON PREVALENCE OF DILATED CARDIOMYOPATHY IN CHRONIC ALCOHOLICS

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MEDICAL UNIVERSITY

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CERTIFICATE

This is to certify that the dissertation title "A STUDY ON PREVALENCE OF DILATED CARDIOMYOPATHY IN CHRONIC ALCOHOLICS", is a bonafide work done by Dr.P.N.RAJESWARAN. It is a regular systematic study done under my guidance and supervision during the period June 2004 to October 2005 and submitted for the ensuring M.D.Branch.I. General Medicine examination September 2006 of Tamilnadu Dr.M.G.R.,Medical University, Chennai.

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DECLARATION

I solemnly declare that dissertation "A STUDY ON PREVALENCE OF DILATED CARDIOMYOPATHY IN CHRONIC ALCOHOLICS", was done by me at Coimbatore Medical College and Hospital during the period June 2004 to October 2005 under the guidance and supervision of Prof. Dr.M.Ramasamy.

I also declare that this bonafide work or part of this work was not submitted by me or any others for any award, degree, diploma to any university or board aided in India and abroad.

The dissertation is submitted to the Tamilnadu Dr.M.G.R., University towards, the partial fulfillment of the requirement for the award of M.D.Degree Branch - I in General Medicine.

Place :

Date :

CONTENTS

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INTRODUCTION

Chronic Alcoholism is rampant in Western Societies. But the alarming feature is that it is also rising in epidemic proportions in developing countries including our country. The main problem with chronic alcoholism is that many of its effects on body systems are usually irreversible. (45).

(eg) Alcoholic Cardiomyopathy (in late stages)

Korsakoff's Syndrome

Atrophy of gastric cells

Cirrhosis of liver

Chronic pancreatitis etc.

As mentioned earlier this study is concentrated mainly around the incidence of dilated cardiomyopathy in chronic alcoholics which manifests as increase in dimension of all four chambers of heart as well as congestive cardiac failure.

However, the hypercontractility of heart muscle seen in earlier stages is usually reversible. Moreover, small doses of alcohol taken daily with adequate diet is thought to be beneficial to heart as it decreases the incidence of cardiovascular death, perhaps by decreasing the incidence of coronary artery and heart disease through its actions on HDL cholesterol and changes in clotting mechanism (23).

But it is very difficult to control or regularize the daily intake of alcohol in people and moreover even low doses of alcohol if taken for a long time is found to increase the cardiovascular morbidity definitely by (8)

1. Depressing myocardial contractility
2. Increasing the heart rate
3. Increased cardiac oxygen consumption
4. Increased incidence of arrhythmias
5. Changes in blood pressure (usually increase)

So if we consider the overall effects of alcohol on heart, various studies clearly establish that, whatever the benefits alcohol offer to heart only minimal and that too transient and questionable whereas the evils it imparts are

unquestionably very high (8). So this study is done to highlight only one of the many evil effects of alcohol i.e. on the cardiac muscle.

AIM OF THE STUDY

The Aim of this study is to find the prevalence of dilated cardiomyopathy in chronic alcoholics and to analyse the cases in detail.

REVIEW OF LITERATURE

Report of the 1995 WHO/International Society and Federation of Cardiology on classification of cardiomyopathies (1)

Dilated cardiomyopathy

Hypertrophic cardiomyopathy

Restrictive cardiomyopathy

Arrhythemogenic right ventricular cardiomyopathy

Unclassified cardiomyopathy

- **Specific cardiomyopathies**

Ischemic cardiomyopathy

Valvular cardiomyopathy

Hypertensive cardiomyopathy

Inflammatory cardiomyopathy

General Systemic Disease

Muscular dystrophies

Neuromuscular disorders

Sensitivity and toxic reactions

Peripartal cardiomyopathy

Etiologic Classification of Cardiomyothies (3)

I. Infective/inflammatory

Idiopathic Iymphocytic Myocarditis

Peripartum myocarditis

Eosinophilic myocarditis

Giant-cell myocarditis

Viral myocarditis

Rickettsial myocarditis

Bacterial myocarditis

Mycobacterial heart disease

Spirochetal heart disease

Fungal myocarditis

Protozoal myocarditis

Metazoal myocarditis

Helminthic myocarditis

Chemical of drug hypersensitivity

Autoimmune myocarditis.

II. Metabolic

A. Endocrine

1. Thyroid disease

Thyrotoxicosis

Hypothyroidism

2. Pheochromocytoma

3. Acromegaly

4. Diabetes mellitus

5. Carcinoid heart disease

B. Uremia

C. Hyperoxaluria

D. Gout

E. Storage disease and infiltrative processes

1. Lysosomal storage diseases

GMI gangliosidosis

Tay-Sachs disease and variants

Sandhoff’s disease

Niemann – Pick disease

Gaucher’s Disease

Farbry’s Disesase

Fucosidosis

Hurler’s syndrome

Schele’s syndrome

Hunter’s Syndrome

Sanfilippo’s syndrome

Morquio’s syndrome

Maroteaux-Lamy syndrome

2. Glycogen storage disease

Pompe’s diseas

Cori’s disease

Andersen’s disease

Dominantly inherited cardioskeletal myopathy

with Iysosomal glycogen’ storage and

normal acid maltase levels

3. Refsum’s disease

4. Hand-Schuller-Christian syndrome

5. Adopositos cordis

6. Hemochromatosis

F. Deficiencies

1. Electrolyte

Hypocalcemia

Hypophosphatemia

2. Nutritional

Kwashiorkor

Beriberi

Pellagra

Scurvy

Selenium

Carnitine

III. Amyloid

AL (primary amyloid, myeloma-associated amyloid)

AA (secondary amyloid, familial Mediterranean fever-associated amyloid)

AF (familial amyloid)

SSA (senile systemic amyloid, senile systemic amyloid)

IAA (atrial amyloid)

IV. General system disorders

A. Collagen vascular

(connective tissue)

Systemic lupus erythematosus

Polyarteritis nodosa

Rheumatoid arthritis

Scleroderma

Dermatomyositis

Whipple’s disease

Kawasaki’s disease

B. Sarcoidosis

C. Neoplastic

V. Muscular dystrophies, myopathies and neuromuscular

Disorders

A. Muscular dystrophies

Duchenne’smuscular dystrophy

Becker’s muscular dystrophy

Myotonic dystrophy

Facioscapulohumeral muscular dystrophy

Limb girdle dystrophy

Scapuloperoneal dystrophy, including

Emery - Dreifuss muscular dystrophy

Congenital muscular dystrophy

Distal muscular dystrophy

B. Congential myopathies

Central-core disease

Desmin myopathy

Multicore myopathy

Nemaline myopathy

Myotubular myopathy (centronuclear)

Congenital fiber-type disproportion

Barth's syndrome

McLeod's syndrome

Bethlem's syndrome

C. Mitochondrial myopathies, including

Kearns-Sayre syndrome

D. Neuromuscular disorders, Friedreich's ataxia

VI. Toxicity, hypersensitivity, and physical agent effects

A. Toxic effects

1. Caused by drugs, heavy metals,

and chemical agents

Alcohol (ethyl)

Amphetamine/methamphetamine

Anthracyclines

Antidepressants

Antimony

Arsenic

Arsine gas

Carbon monoxide

Catecholamines

Chloroquine

Cobalt

Cocaine

Cyclophosphamide

Emetine

5-Fluorouracil

Hydrocarbons

Interferon

Lead

Lithium

Mercury

Methysergide

Paracetamol

Phenothiazines

Phosphorus

Reserpine

2. Caused by scorpions, spiders,

arthropods, and snakes

Scorpions

Arthropods

Black widow spider

Snakes

B. Hypersensitivity reactions

Acetazolamide

Amitriptyline

Amphotericin B

Ampicillin

Carbamazepine

Chlorthalidone

Hydrochlorothiazide

Indomethacin

Isoniazid

Methyldopa

Oxyphenbutazone

Para-aminosalicylic acid

Penicillin

Phenindione

Phenylbutazone

Phenytoin

Streptomycin

Sulfadiazine

Sulfisoxazole

Sulfonylureas

Tetracycline

C. Physical agents

Heat

Hypothermia

Radiation

VII. Miscellaneous

Peripartum heart disease

Tachycardia-induced cardiomyopathy

Ectodermal dysplasia-associated cardiomyopathy

Idiopathic endocardial fibrosis

Endocardial fibroelastosis

Infantile cardiomyopathy

Arrhythmogenic right ventricular dysplasia

Carbohydrate-deficient blood glycoprotein syndrome

Simpson-Golabi-Benhmel syndrome

Isolated ventricular noncompaction syndrome

Myoadenylate deaminase deficiency.

Dilated cardiomyopathy:

It is defined as primary disease of myocardium with dilatation of all four chambers of the heart ultimately producing gradual development of congestive heart failure (1).

Alcoholic cardiomyopathy:

Strictly speaking cardiomyopathies are idiopathic whenever a demonstrable cause is found (eg. alcohol) it should be termed as specific (eg. alcoholic) heart muscle disease. However, the exact mechanism by which alcohol causes myocardial damage is still unknown and hence it continues to be regarded as possible basis or associated factor in dilated cardiomyopathy (45).

There has been speculation that alcohol caused myocardial damage only through dietary deficiencies but it is now clear that alcoholic cardiomyopathy occurs in the absence of nutritional deficiency (1).

Incidence:

Six per one lakh in the general population (per year)

Prevalence:

Twenty per one lakh in the general population.

Prevalence in chronic alcoholics:

The SEYCHELLES STUDY, conducted by VICTORIA HOSPITAL, SEYCHELLES reveals a prevalence of alcohol related cardiomyopathy in 20% of chronic alcoholics.

Pharmacology of Ethanol (46) :

It is a weakly charged molecule that moves easily through all membrane.

Absorbtion : From mucosa of mouth and oesophagus (in small amount) and from stomach and intestine (large amounts). Rate of absorbtion increases with rapid gastric emptying.

The absence of proteins, fat or carbohydrates, dilution to about 20% and carbonation.

Congeners:

These are substances found in alcohol beverages and may contribute to body damage with heavy drinking. They decrease the absorbtion of alcohol.

(eg.) Low molecular weight alcohols (methanol)

aldehydes,

esters,

histamine phenol,

tannins,

Iron,

Lead

and importantly cobalt.

Metabolism:

1. 2 - 10% of ingested alcohol is directly excreted through lungs, urine, sweat.
2. A major portion (about 80%) is metabolized in cell cytosol via alcohol dehydrogenase.

Ethyl alcohol $\xrightarrow[NAD]{}$ Acetaldehyde

Acetaldehyde $\xrightarrow[NAD]{}$ Acetic acid

3. 10% of the ingested alcohol is metabolized by microsomal ethanol oxidising system of microsomes of smooth endoplasmic reticulum.

Increased activity of this system can be induced after repeated exposure to ethanol.

Both the pathways (2) and (3) require Nicotinamide adenine dinucleotide as cofactor. And it is the increased ratio of this reduced cofactor NADH to NAD i.e. NADH : NAD is responsible for many of the metabolic dearrangements observed after alcoholic ingestion.

IDENTIFICATION OF AN ALCOHOLIC: (28)

1. History : Either from the patient or from his relatives.
2. Blood alcohol estimation
3. High normal or slightly elevated mean corpuscular volume
4. Gamma glutamyl transferase : 35-40 units or more
5. serum uric acid : 7 mg /100 ml / or more
6. Triglycerides : 180 mg per 100 ml or more
7. Mild and fluctuating levels of hypertension,
   repeated infections,
   un-explained cardiac arrhythmias,
   cutaneous stigmata,

bilateral parotid swelling etc.

Mechanism by which alcohol produces cardiac damage (33).

The cardiotoxicity is due to an intrinsic effect of alcohol rather than to malnutrition or co-toxicity. The various mechanisms proposed are:

a. Interference with number of cellular functions by acetaldehyde that involve (13).

- Transport and binding of calcium
- Mitochondrial respiration
- Myocardial lipid metabolism
- Myocardial protein synthesis
- Myofibrillar ATP ase

b. Electrolyte imbalance at cellular level producing secondary toxicity (12).

Chronic alcohol ingestion induces deficiency of

myocardial magnesium, potassium and zinc.

Immunological mechanisms (39)

Antibodies to nerve fibres of the myocardium could be demonstrated in the presence of T-lymphocyte deficiency and hightitres of antibodies to EB virus in patients with alcoholic cardiomyopathy.

Recently, a non – oxidative pathway for the metabolism of alcohol in several organ system including the heart has been described. Non esterified fatty acids on esterified with ethanol to produce fatty acid ethyl esters (FAEE). These molecules can accumulate in mitochondria and impair cellular function. Fatty acid ethyl esters are synthesized at high rates in the heart owing to the lack of oxidative ethanol metabolism in this organs.(3).

Pathology:

A. Macroscopic Picture (10) (45)

Enlargement and dilatation of all four cardiac chambers. The ventricles are more dilated than atria while thickness of the ventricular wall is increased in some cases the degree of hypertrophy is often inadequate for the severe dilation present. The cardiac valves are intrinsically normal and intracavitary thrombi particularly in the ventricular apex are common. The coronary arteries are usually normal.

B. Microscopic Picture (10):

It reveals extensive areas of interstitial and perivascular fibrosis, occasionally associated with calcification, cellular infiltration is not prominent. However, interstitial fibrosis cellular hypertrophy, mycocardial cell degeneration are seen in many cases.

C. Electron Microscopy (13)

It reveals abnormal mitochondria, swelling and loss of cristae, increased glycogen accumulation, fatty change etc.

None of the features is either specific or sensitive. However, pathological examination aids in ruling out other causes of dilated cardiomyopathies or myocarditis.

Because not all alcoholics develop cardiomyopathy, the relationship between the development of cardiac dysfunction and dose of alcohol is complex and probably multifactorial. There appears to be a genetic predisposition to the development of cardiomyopathy (5) because with the DD genotype of the

angiotensin converting enzyme are 16 times more likely to develop cardiac dysfunction than those without:

Symptoms of cardiac damage (1) (3)

1. Dyspnea on exertion
2. Fatigue
3. Orthopnea
4. Paroxysmal nocturnal dyspnea
5. Edema
6. Palpitation
7. Cough
8. Chestpain

Signs: (1) (37)

These are due to cardiac failure.

1. Cardiomegaly
2. Small pulse pressure
3. Elevated JVP.
4. 3^{rd} and 4^{th} heart sounds
5. Murmurs of tricuspid regurgitation and mitral regurgitation.

6. Edema
7. Hepatomegaly
8. Ascitis
9. Basal crepitation in lungs
10. Pleural effusion:

In addition signs of chronic alcoholism like

1. Testicular atrophy
2. Spider naevi
3. Palmar erythema
4. Obesity
5. Dupuytrens contracture

Laboratory investigations:

I. X ray Chest – PA view

Cardiomegaly

Pulmonary venous hypertension.

II. Electrocardiography (1) (3)

1. Non Specific ST, T Changes
2. Hypertrophy and enlargement of all four chambers.
3. Intraventricular conduction defects

4. Poor progression of R wave across precordium.

5. Various arrhythmias

   i. Sinus tachycardia

   ii. Atrial fibrillation

   iii. Paroxysmal atrial tachycardia

   iv. Ventricular ectopics

   v. Ventricular tachycardia

   vi. A.V.conduction disturbance

6. Q waves may be seen when there is extensive left ventricular fibrosis without diffuse myocardial infarction.

III. Echocardiogram / Doppler studies (1) (9) (17) (18)

i. Size of cardiac chambers and ventricular cavity.

ii. Thickness of the chambers.

iii. End diastolic and systolic volume

iv. Ejection fraction

v. Fractional shortening

vi. Mitral regurgitation and tricuspid regurgitation.

vii. Thrombus inside the chambers.

viii. Pulmonary hypertension

ix Pericardial effusion

x. Excluding other structural abnormalities

IV. Hemodynamic Studies (1) (6)

a. Cardiac output

b. Left and right ventricular enddiastolic pressure.

c. Left and right atrial pressure.

d. Pulmonary capillary wedge pressure.

e. Central venous pressure.

V. Angiographic Studies: (1) (14) (15).

a. Dilated and diffusely hypo kinetic left ventricle.

b. Mitral and Tricuspid regurgitation.

c. Normal coronary arteries.

d. Dilatation of other chambers.

VI. Radionuclide studies (22)

Left ventricular dilatation and dysfunction.

VII. Transvenous Endomyocardial Biopsy. (10) (45)

It is important mainly in ruling out myocarditis and pin pointing the etiology of cardiomyopathy. But this interventional procedure has its own harmful effects.

Treatment: (1) (2) (3)

1. Absolute abstinence from alcohol consumption before severe heart failure has developed may halt the progression or even reverse the course of the disease.
2. Correction of thiamine deficiency if it is there.
3. Good nutritious diet with adequate supplementation of vitamins and minerals.
4. Treatment of infections if present.
5. Treatment of cardiac failure.
    - Avoidance of strenuous exertion
    - Salt restriction
    - Digoxin
    - Diurtics
    - ACE inhibitors
    - Nitrates
    - To decrease the incidence of thromboembolism antico-agulants may be added.

Treatment of Arrhythmias

- Specific drugs
- Surgical interruption or implantation of an automatic internal defibrillator.

7. Cardiomyo plasty (19)

With flaps raised from lattismus dorsi muscle with intact neuromuscular bundle.

8. Cardiac Transplantation

This should be considered in any patient who has advanced stage of the disease and who is refractory to treatment and who have no contra indications to the procedure.

Prognosis in Alcoholic Cardiomyopathy (11) (32)

It is poor in patients who continue to drink, particularly if they have been symptomatic for a long period. In one study 80 % of such patients die within in a 3 years period. In the overall population of patients with alcoholic cardiomyopathy between 40 – 50% die within 3 – 6 years period.

PRECLINICAL ALCOHOLIC CARDIOMYOPATHY (20)

It is a well known entity in chronic alcoholics who are usually 40 years or less of age. It is noted in the autopsy findings of those who died due to some other cause. However extensive studies were under taken at cardiology section SEATTLE VETERANS AFFAIRS MEDICAL CENTRE, WASHINGTONE on chronic alcoholics confirmed the existence of such an entity in chronic alcoholics and also observed that if alcohol consumption is stopped at this stage, progression of this to clinically obvious cardiomyopathy can be prevented.

Doppler echocardiography is quite sensitive in evaluating diastolic dysfunction even before the development of clinically evident dysfunction. Patients had,

Prolonged relaxation time

Decrease in peak early diastolic velocity

Slower acceleration of early diastolic flow

Higher atrial to early peak velocity (2)

Other cardiotoxic effects of ethylalcohol apart from cardiomyopathy are.

1. LV Dysfunction without frank dilated cardiomyopathy (37)

2. Arrhythmias including holiday heart syndrome (41)(35)

3. Prolonged QT syndrome (35)

4. Hypertension

5. Sudden cardiac death (43)

6. Cardiac autonomic neuropathy

7. Beri Beri heart disease

8. Fatty myocardial infiltration

9. Pericardial effusions

10. Fetal Alcohol syndrome with a variety of cardiac lesions (usually V.S.D) in children born to mothers who are chronic alcoholics.

DEFINITIONS

1. The Natural council of Alcoholism, united status and American Medical Society on Alcoholism in collaboration with WHO Defines Chronic alcoholism as a

   "Pathological state resulting from habitual use of alcohol in toxic amounts"

2. The original version of the fourth Diagnostic and statistical manual of the American psychiatric Association (DSM IV) divide chronic Alcoholism into two types.

   A. Alcohol Abuse : It indicates psychological dependence.
   B. Alcohol Dependence: It indicates mainly physical dependence, tolerance, increased clearance and withdrawal symptoms on abstinence.

DEFINITION OF CHRONIC ALCOHOLISM WITH REFERENCE TO HEART

Since, non of the above definitions is useful from a clinical point of view, observations made by various experts in the field of cardiology is given below. These definitions are useful both epidemiologically and clinically.

The various observations are:

1. Consumption of 80 grams or more of alcohol per day for a period of 8 – 10 years is known to be associated with cardiomyopathy.

   (Lelbach, Pequignat – **Recent Advances in Medicine***)*

2. Consumption of 100 grams of more of alcohol per day for a period of more than 10 years will injure the heart muscle and lead to cardiomyopathy.

   (Charles Friedberg **- Diseases of heart***)*

3. Cardiomyopathy develops in persons drinking 80 grams of alcohol per day for atleast ten years .

   (Michael H.Crawford - **Cardiology***)*

4. Most patients developing alcoholic cardiomypathy have been drinking more than 80 grams of alcohol per day for more than 5 years.

   (James T.Willerson, Jay – N.Cohn-**Cardiovascular Medicine***)*

5. Frequent Binjing without heavy daily consumption may also be sufficient to produce cardiomyopathies.

   (Lynne Warnar Stevenson – **Cecil Text Book of Medicine***)*

6. Criteria used to define heavy chronic alcohol use have included such estimates as the use of 125 ml/day of alcohol or 30 - 50% daily calories derived from alcohol for a minimum of 10 years.

   (Seon P.Pinney, Donna M.Moncine **– Hursts' The Heart***)*

7. 8 g of alcohol = ½ pint of beer = 1 Single measure (25 ml spirit =1glass of wine

   (Kumar & Clack - Clinical Medicine)

ALCOHOL CONTENT OF CERTAIN COMMON BEVERAGES

Beverages	Content of Alcohol
Beer	2.6%
Whisky	40-50%
Rum	51-59%

Gin	40%
Brandy	40 - 50%
Country Arrack	60 – 70%

(Toxicology – Modi)

WORKING DEFINITION OF CHRONIC ALCOHOLISM ADOPTED IN THIS STUDY.

So, after studying the above observations a working definition was adopted in this study. It defines chronic alcoholism as.

"*Those who consume more than 80 g of Alcohol per day in atleast 6 days per week for a period of atleast ten years*".

MATERIALS AND METHODS

Period of study : From June 2004 to October 2005

Total number of cases studied: Seventy Cases.

Case Selection:

1. Patients for this study were selected at random in General medical wards and general and cardiology OP department in Coimbatore Medical college hospital.
2. Only patients who satisfy the working definition are included.
3. Patients with the following conditions are omitted.
    a. Patients with history of Ischemic heart disease, ECG changes suggestive of Ischemic heart disease.
    b. Patients with the following medical problems.

        Systemic hypertension

        Diabetes mellitus

        Bronchial Asthma

        Renal disease

        Gross nutrional disorders

        Liver disease

        Hyper calcemic states

        Hyper cholesterolemia

        Thyroid disease and other endocrine problems.

5. Only males are chosen because chronic alcoholism is not commonly seen in woman in our country and also to avoid the possible unnecessary interference of hormonal factors in this study.
6. Patients from Coimbatore City only are in this study in order to avoid the possibility of any environmental or geographic influences over this study.
7. Patients belonging to the same socio economic class are chosen with a mean per capita monthly income of 500 Rupees.
8. Patients who are on long term drugs are avoided.
9. It is very difficult to get pure chronic alcoholics without smoking, so heavy smokers are included.

(None of the studies mention relation of cigarette smoking and incidence of cardiomyopathy).

CASE DEFINITION

1. All patients should satisfy the working definition of chronic alcoholism.

2. Cases should have evidence of dilated cardiomyopathy like.

I. Clinical Examination: This usually shows :

a. Down and outward shift of apical impulse.

b. Diffuse, hypokinetic apical impulse.

c. Evidence of functional tricupid regurgitation and /or Mitral regurgitation.

d. Third or Fourth heart sounds.

II. X ray Chest – PA view

Cardiomegaly

Pulmonary venous hypertension.

III. Electrocardiography (1) (3)

- Non Specific ST, T Changes
- Hypertrophy and enlargement of all chambers.
- Intraventricular conduction defects
- Poor progression of R wave across precordium.

Various arrhythmias

i. Sinus tachycardia

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Q waves may be seen when there is extensive left ventricular fibrosis without diffuse myocardial Infarction.

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d. Pulmonary capillary wedge pressure.

e. Central venous pressure.

VI. Angiographic Studies: (1) (14) (15).

e. Dilated and diffusely hypo kinetic left ventricle.

f. Mitral and Tricuspid regurgitation.

g. Normal coronary arteries.

h. Dilatation of other chambers.

VII. Radionuclide studies (22)

Left ventricular dilatation and dysfunction.

VIII. Transvenous Endomyocardial Biopsy. (10) (45)

Various studies clearly mention that endomyocardial biopsy does not yield characterstic microscopic picture which may be of use in diagnosing dilated cardiomyopathy or pin pointing alcohol as the etiological agent.

Various authorities like P.C.Manoria, Alioa.L.P., Mckenna J.State that 2 D echo itself would be sufficient in confirming the diagnosis of Dilated cardiomyopathy.

Moreover endomyocardial biopsy is hazardous in these patients. As it is an invasive procedure and in these patients with dilated hearts it may precipitate fatal arrhythmias.

Facilities for the procedure V – VIII are not available in our hospital so they were not done.

MODE OF STUDY

Careful questioning was done and various symptoms relevant to the cardiovascular symptoms were elicited and the observations are listed.

All the patients are carefully examined clinically various valuable relevant signs are noted.

The following investigations were performed on all patients.

1. Total WBC count.
2. Differential count
3. Erythrocyte sedimentation rate.
4. Hemoglobin content.
5. Urine analysis
6. Blood urea and Serum creatinine
7. Blood sugar
8. Lipid profile
9. Liver function test
10. Thyroid function test.
11. Serum Electrolytes
12. X ray chest
13. Electro cardiography
14. Echocardiogram / Doppler

Followup: All cases are followed up for one month and a repeat echocardiogram was done on patients who initially showed dilated cardiomyopathy. This is done to ruleout the possibility of any acute myocarditis which might have caused dilated heart chambers.

OBSERVATIONS

The following are observations made in my study

Age Group with prevalence of cardiomyopathies

Age Group	No. of cases	No. of cases of	%

(years)	studied	cardiomyopathies	
30-40	5	0	0
40-50	28	4	14%
50-60	20	4	20%
60-70	17	4	24%

This table clearly shows as the mean age increases and the duration of exposure to alcohol increases the risk of developing cardiomyopathy also increases.

CLINICAL FEATURES

Symptoms	Present in	%
Cough	62	89%
Exertional Dyspnea	54	77%

Precordial Pain	8	11%
Palpitation	36	50%
Fatigue	ALL	100%
Syncopial Attacks	3	4%
Swelling of legs	20	30%
Right hypochondrial pain	38	54%
Hemptysis	Nil	0%
Orthopnea/PND	9	14%

This apparent discrepancy between the no. of cases of DCM and presence of symptoms in large no. of patients may be due to the fact that most of them are nonspecific for example edema occurs in a variety of setting and cough may be due to respiratory tract infection and fatigue is obviously a nonspecific symptom.

GENERAL EXAMINATION

Signs	**Present in**	**%**
Obesity	42	60%
Undernourishment	None	0%
Anemia	None	0%

Jaundice	None	0%
Clubbing	None	0%
Cycosis	7	10%
Cutaneous stigmata of chronic alcoholism	15	20%
Elevated JVP	6	9%

It is to be noted that from the above tables that certain findings such as hypertension, shock, anemia, jaundice etc. are not present in this study. This is because cases are selected to avoid unnecessary influence of extraneous forces over the out come of study.

SYSTEMIC EXAMINATION

Signs	**Present in**	**%**
Apical Impulse (Down and Outward Shift)	12	17%
Auscultatory Signs of MR/TR	10	14%

S3/S4 Gallop	6	9%
Hepatomegaly a)Tender b)Non Tender	 6 16	 9% 21%
Basal rales	14	20%
Small Volume pulse	10	14%
Hypertension	0	0%

It is to be noted from above table that certain findings like non tender hepatomegaly and basal crepts are non specific. As non tender hypatomegaly in alcoholics may be due to fatty infiltration of liver and basal crepts may be due to respiratory infection apart from cardiac failure. Patients with hypertension are already excluded in this study because of unnecessary bias.

ECG

Type of Tachyarrhythmias	**Present in**	**%**
Ventricular ectopics	2	3%
Atrial Fibrillation	4	6%

Supraventricular tachycardia	1	1.5%
Non sustained ventricular tachycardia	1	1.5%

Type of block	**No. of cases**	**%**
First degree block	1	1.5%
Complete heart block	1	1.5%
LBBB	2	3%
LAHB	1	1.5%

CHEST X RAY

Findings	**Present in**	**%**
Cardiomyopathy	12	17%
Pulmonary hypertension	6	9%

Alveolar edema	2	3%

It is to be noted from the above table that pulmonary hypertension and alveolar edema are present in patients with cardiac failure and particularly alveolar edema in patients with severe heart failure.

ECHO/DOPPLER

Findings	**Present in**	**%**
Enlargement of all 4 chambers	12	17%
Increased end systolic volume	8	11%

Ejection fraction < 50	8	11%
Thrombus in chambers	1	1.5%
Mitral and tricuspid regurgitation	10	14%

Above table gives information that although 12 patients are known to have DCM only 8 patients have cardiac failure as evidenced by their echocardiogram finding, increased end systolic volume and ejection fraction <50%. One patient had thrombus in left ventricle.

DISCUSSION

In this study of seventy cases of chronic alcoholism, twelve cases of dilated cardiomyopathy were noted. Though there are numerous other causes of

dilated cardiomyopathy, certain factors are against those (obviously peripartum dilated cardiomyopathy would not interfere in this study because only males are taken into account). Patient with ischemic heart disease are not included in this study. Patients on long term drug therapy (any drug) and toxin exposure are not taken, so the problem of drug factor is also ruled out.

Glycogen storage diseases are unlikely in this study as they are seen almost exclusively in paediatric age group whereas in this study all the patients are above thirty. Muscular dystrophies are ruled as there were no signs and symptoms of muscle weakness in any patients. Infections also cause dilated cardiomyopathy notably viral infections. Only endomyocardial biopsy can distinguish between these two.

But again infections causing myocarditis are more common in younger age group and the course is usually sudden with rapid progression to cardiac failure whereas alcoholic cardiomyopathy has a typical insidious onset with slow progression of cardiac failure which is usually very difficult to control with drugs.

So among the left out causes of dilated cardiomyopathy the granulomas and connective tissue disease are unlikely as they all have systemic

manifestations. So also are the deficiency states and metabolic problems like hypo / hyperthyroidism. So only two are now left with. They are the idiopathic and familial varieties. They are quiet rare and their etiological contribution is very difficult to prove.

In this study of chronic alcoholism the prevalence of dilated cardiomyopathy is 17% which goes roughly in parallel with the SEYCHELLES STUDY conducted by the VICTORIA HOSPITAL, SEYCHELLES which is till to date largest study of its kind in the world (it gives a prevalence of 20% among chronic alcoholics.

Study	Prevalence
SEYCHELLES STUDY	20%
OUR STUDY	17%

Taking age group into consideration as the mean age increases, the duration of exposure to alcohol increases, the risk also increases which is reflected in our study also.

Taking symptoms in to consideration. SEGEL J.P.et al fees that cough, dyspnea on exertion, fatigue are common and early manifestations of this disease. In our study also.

Cough (89%)

Dyspnea on exertion (77%)

Fatigue (100%)

are commonly encounted.

Regarding mitral regurgitation and tricuspid regurgitation murmurs due to ventricular dilation, observation made by SCHLANT R. state that they are found in 75% of patients with Dilated cardiomyopathy. It roughly correlates with our study also.

STUDY	MR/TR
SCHLANT R.STUDY	75%

OUR STUDY	80%

Arrhythmias and conduction blocks are infact known to occur very commonly in acute alcoholic intoxication also. This is called Holiday heart syndrome. Usually these patients will not have evidence of structural heart disease.

Dilated cardiomyopathy per se itself lead on to various arrhythmias. FAUDAL H.CHENG.et al pointed that 10% of chronic alcoholics have conduction disturbances. In our study it is about 7%.

STUDY	CONDUCTION DISTURBANCES
FAUDAL H.CHENG.et al	10%
OUR STUDY	7%

CONCLUSION

The main purpose of this study is to high light the prevalence of dilated cardiomyopathy in chronic alcoholics. In this study of seventy cases of chronic alcoholics dilated cardiomyopathy was noted in twelve patient this amount to prevalence of 17%.

This study results roughly correlate with the SEYCHELLES STUDY conducted by VICTORIA HOPITAL. In this study the prevalence was 20%.

This prevalence rate is definitely high when compared to the prevalence of alcoholic dilated cardiomyopathy in normal population which is twenty per one lakh. This is due to selection of cases who are chronic alcoholics with symptoms relevant to cardiovascular system.

SUMMARY

The observations noted in this study are,

Out of 70 chronic alcoholics studied 12 patients found to have dilated cardiomyopathy.

Out of these 12 patients of dilated cardiomyopathy, 8 patients found to have cardiac failure, which amounts to 70%.

Tachy arrhythmias noted in 8 patients out of 12 patients, which is 70%

Conduction disturbances in 4 patients,out of 12 patients, which is 30%

Atrio ventricular valvular regurgitation in 10 patients out of 12 patients, which is 80%.

Left ventricular thrombus in one patient.

ANNEXURE

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PERFORMA

Name Age Sex

Address Occupation

Complaints

Cough Syncope

Exertional Dyspnoea Swelling of legs

Precordial pain Abdoman pain

Palpitation Hemoptysis

Fatigue Orthopnoea /PND

Past History Hypertension Diabetesmellitus

CAHD Tuberculosis

Diet Veg Non Veg. Mixed

Smoker

Alcoholic Years Frequency/week

Extramarrital contact

Family History:- Any persons in family with similar ill

Examination

General -Examination

Nourishment Thin built obese

Anemia Jaundice

Clubbing Pedaledema

Cyanosis JVP

PR Rate Rhythm Peripheral pulse

BP RR

Cutaneous stigmata of chronic alcoholism

Systemic Examination

CVS

Apical impulse position character

Auscultatory signs of MR/TR

S3/S4 gallop

RS

Breath Sounds

Added Sounds

Abdomen

Organomegaly

Investigations

CHG ESR Hb%

Blood Urea Sr. Creatinine

Blood Sugar (F)	Sr. Cholesterol
LFT	TFT
Sr. Electrolytes	
Urine Analysis	
X-ray Chest – PA	
ECG	
Echo/ Doppler	

MASTER CHART

Name	Age	No.	U	Cr	S	TFT	S.E	ECG	X – ray	ECHO D	EF	MR/TR
Abdul	47	3009	30	0.8	96	N	N	N	N	--	N	--
Anganna	55	11126	30	1	95	N	N	N	N	--	N	--
Annavi	42	51800	34	0.9	104	N	N	AF	CM	+	< 50%	--
Arunachalam	44	2980	32	0.9	92	N	N	N	N	--	N	--
Asf Bai	53	7120	32	1	101	N	N	N	N	--	N	--
Ayyavu	62	90021	28	1.1	100	N	N	N	N	--	N	--
Babu	36	70012	26	1.1	98	N	N	N	N	--	N	--
Bairavan	40	51899	30	1.2	94	N	N	N	N	--	N	--
Balan	45	67778	28	1.1	97	N	N	CHB	CM	+	<50%	--
Balasubramani	35	31899	28	0.9	98	N	N	N	N	--	N	--
Chandran	45	81181	30	0.8	103	N	N	N	N	--	N	--
Chandrasekar	42	4002	32	0.9	104	N	N	N	N	--	N	--
Chelliah	59	85001	50	1.8	101	N	N	LBBB	CM,PVH	+	<50%	+
Chenniappan	50	5881	35	1	102	N	N	N	N	--	N	--
Chinnan	70	4824	32	1.2	95	N	N	N	N	--	N	--
Danel	39	11180	26	0.8	99	N	N	N	N	--	N	--
Devegan	47	21876	32	1	96	N	N	N	N	--	N	--
Dhanavel	60	11222	28	0.9	100	N	N	N	N	--	N	--
Dhandapani	51	30809	28	0.8	95	N	N	N	N	--	N	--
Ebenesan	43	99336	28	0.8	89	N	N	N	N	--	N	--
Erudayaraj	50	66331	35	1.2	96	N	N	N	N	--	N	--
Erulappan	65	90036	50	2.0	82	N	N	SVT	CM,PVH	+	<50%	+
Esaki	70	55118	32	1.1	92	N	N	N	N	--	N	--
Farooq	41	61182	32	0.9	78	N	N	N	N	--	N	--
Gandhi	67	7645	40	1.1	96	N	N	N	N	--	N	--
Ganesan	40	54321	28	0.8	99	N	N	N	N	--	N	--
Gnanam	58	21218	23	0.7	75	N	N	N	N	--	N	--
Gopal	48	80002	30	1	80	N	N	N	N	--	N	--
Govindhan	56	5289	32	1.1	82	N	N	N	N	--	N	--
Gundumani	60	20088	48	2	102	N	N	NSVT	CM,PVH	+	<50%	+
Haider Ali	51	236	50	0.5	103	N	N	AF	N	--	N	--
Haniffa	67	33311	32	1	98	N	N	N	N	--	N	--
Hariharan	49	78118	35	1.1	90	N	N	N	N	--	N	--
Ismail	65	18321	28	0.9	89	N	N	N	N	--	N	--
Iyyadurai	59	71809	34	1.1	86	N	N	N	N	--	N	--
Iyyappan	45	1732	26	0.8	83	N	N	VPC	CM	+	N	+
Iyyavu	46	12888	36	1.2	92	N	N	N	N	--	N	--
Jayan	52	12089	36	1.2	90	N	N	N	N	--	N	--
Kali	48	991	40	1.1	83	N	N	N	N	--	N	--
Kandasamy	49	3381	28	0.8	90	N	N	N	N	--	N	--
Karuppaiya	60	1990	28	0.9	90	N	N	N	N	--	N	--
Karuppan	57	204	36	0.9	87	N	N	N	N	--	N	--
Karupusamy	61	41899	48	2.1	95	N	N	AF	CM,PVH	+	<50%	+

Kesavan	43	2140	30	0.8	98	N	N	N	N	--	N	--
Kittan	58	521	26	0.8	92	N	N	N	N	--	N	--
Kondan	67	806	30	0.9	101	N	N	N	N	--	N	--
Mani	51	2980	33	1.1	89	N	N	VPC	CM	+	N	+
Maran	46	70082	30	0.9	82	N	N	N	N	--	N	--
Mariyappan	65	65812	24	0.9	99	N	N	N	N	--	N	--
Maruthachalam	64	27792	27	0.8	86	N	N	N	N	--	N	--
Meganathan	46	33009	34	0.9	91	N	N	N	N	--	N	--
Munusamy	69	1192	54	1.8	93	N	N	AF	CM,PVH	+	<40%	+
Murthi	37	6682	32	1.1	105	N	N	N	N	--	N	--
Palanisamy	55	56828	28	1	90	N	N	N	N	--	N	--
Periasamy	57	58271	36	1	92	N	N	N	N	--	N	--
Pitchai	64	500	40	1.2	94	N	N	N	N	--	N	--
Rajan	36	233	24	0.8	98	N	N	N	N	--	N	--
Raman	40	3132	24	0.8	91	N	N	N	N	--	N	--
Ramanan	42	29542	27	0.7	90	N	N	N	N	--	N	--
Ravindren	44	60821	42	1.2	88	N	N	N	N	--	N	--
Rengan	51	55291	26	0.8	86	N	N	N	N	--	N	--
Sabakarim	56	59981	32	1.1	97	N	N	N	N	--	N	--
Saiyed	49	60266	29	1	96	N	N	LAHB	CM	+	N	+
Salesten	46	58291	25	0.8	99	N	N	N	N	--	N	--
Subbiah	51	2281	28	0.9	103	N	N	N	N	--	N	--
Subramaniam	41	62891	32	0.9	96	N	N	N	N	--	N	--
Sundharam	52	6282	54	2	89	N	N	LBBB	CM,PVH	+	<50%	+
Thangaraj	50	54560	34	0.9	84	N	N	CHB	CM	+	N	+
Vashudevan	43	21006	30	0.8	88	N	N	N	N	--	N	--
Velan	45	1191	34	0.9	92	N	N	N	N	--	N	--

A	:	Age
U	:	Blood Urea
CR	:	Serum Creatinine
S	:	Blood Sugar (F)
TFT	:	Thyroid Function Test
SE	:	Serum Electrolytes
AF	:	Atrial Fibrillation
VPC	:	Ventricular ectopics

NSVT	:	Non Sustained Ventricular Tachycardia	PVH	:	Pulmonary Hypertension
CHB	:	Complete Heart Block	D	:	Dilatation of all four chambers
EF	:	Ejection fraction	LBBB	:	Left Bundle Branch Block
MR/TR	:	Mitral and Tricuspid regurgitation	CM	:	Cardio megaly